crispprd 1.0

Abstract

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Grant Number:	5R01NR005281-02
PI Name:	SHERWOOD, ANDREW
PI Title:	ASSOCIATE PROFESSOR
Project Title:	HEART DISEASE IN WOMEN: ESTROGEN EFFECTS ON HEMODYNAMICS

Abstract: *DESCRIPTION: Coronary heart disease (CHD) has now become the leading cause of death in women. The incidence of CHD increases sharply following menopause, while estrogen levels decline. Data from observational studies suggest that higher estrogen levels may protect women from developing CHD. There is also evidence that estrogen may protect women who already have CHD. However, the cardioprotective benefits of estrogen alone may be countered by the addition of progesterone, which is typically included in hormone replacement therapy (HRT). The Heart and Estrogen/Progestin Replacement Study (HERS) recently reported that progesterone-estrogen combination therapy did not protect women with CHD from myocardial infarction and/or death. The mechanisms responsible for estrogen's cardioprotective benefits have not been fully identified. Estrogen replacement improves lipid profiles, but this mechanism alone accounts for only approximately 25 percent of the reduction in risk for mortality. Recent studies have shown that estrogen also appears to improve vascular endothelial function, which is compromised in CHD. Endothelial dysfunction simultaneously reduces myocardial oxygen supply by limiting coronary vasodilation, while increasing demand by elevating systemic vascular resistance. Depending upon the severity of this imbalance, CHD patients may experience myocardial ischemia, myocardial infarction and/or cardiac death. Several studies have shown that stress may exacerbate this pathophysiological imbalance in CHD and trigger acute events. The purpose of the proposed research is to further our understanding of estrogen and estrogen/progesterone's effects on endothelial function and its consequent impact on SVR at rest and during stress. In a randomized double-blind crossover comparison of 60 women with documented CHD and 60 age-matched healthy*

women, we propose to examine the acute effects of estrogen and estrogen/progesterone interventions on SVR at rest and during stress. Vascular endothelial function will be assessed using ultrasound imaging of flow-mediated dilation of the brachial artery. Using recently developed ambulatory monitoring technology, blood pressure, cardiac output and SVR will be monitored non-invasively at rest, during laboratory-based mental stress, and for 24 hours during patients' normal daily routines. By these methods, the proposed research should clarify how HRT may alter risk in CHD through hemodynamic effects that are hypothesized to be secondary to alterations in endothelial function.

Thesaurus Terms:
coronary disorder, estrogen, female, hemodynamics, hormone regulation /control mechanism, hormone therapy, human therapy evaluation, vascular resistance blood pressure, cardiac output, cytoprotection, progesterone clinical research, human subject, ultrasound imaging /scanning, women's health

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